VALSALVA HEMORRHAGIC RETINOPATHY*

BY *Thomas D. Duane*, MD

THAT THE FUNDUS, ESPECIALLY THE RETINA, RESPONDS TO ALTERATIONS OF homeostasis produced by distant trauma has been recognized since shortly after the discovery of the ophthalmoscope.

It is the purpose of this paper to describe a particular form that this retinopathy may assume, Valsalva hemorrhagic retinopathy. An additional purpose is to attempt to put this ophthalmoscopic appearance in proper perspective by presenting probable pathophysiologic mechanisms categorized under the headings of "forward" (arterial) and "backward" (venous) vasculopathies respectively. The goal is to bring order to the interpretation of these entities by demonstrating that the most common picture, Purtscher's retinopathy secondary to distant trauma, is, in all likelihood, a mixture of forward and backward mechanisms operating to various degrees in any given patient.

PATIENT REPORTS

CASE 1

Presented through the courtesy of Edwin U. Keates, MD, of Philadelphia.

This 38-year-old black female was examined in January 1968 with a questionable history of having been hit in the right eye while playing at judo with her boyfriend several days previously. Vision in the right eye was counting fingers at one meter. A slight flare was present in the anterior chamber. The fundus showed patchy, dark red areas throughout the posterior pole, interspersed with some paler areas and fine dark lines radiating from the disc. Vision in the left eye was 6/6 but the fundus showed marked angioid streaks with no hemorrhages. Systemic examination was normal. The FTA-ABS and VDRL tests were non-reactive. Roentgenograms of the skull and long bones were within normal limits. There was no evidence of sickle cell anemia and a skin biopsy from the axilla was interpreted as being positive for pseudoxanthoma elasticum. A diagnosis of bilateral angioid streaks with superim-

*From the Department of Ophthalmology, Jefferson Medical College, Thomas Jefferson University, Philadelphia, Pennsylvania.

TR. AMER. OPHTHAL. SOC., VOL. LXX, 1972

posed traumatic retinopathy of the right eye was established. The mild iritis in the right eye improved at once with local and systemic steroids and with cycloplegics.

In April 1968 the patient was examined again. The vision in the right eye had improved to 6/18. Ophthalmoscopic examination revealed that the hemorrhage had been absorbed. The angioid streak, of course, remained, and there was a small glial scar adjacent to the macula. The left eye was as before.

The patient was seen once again in August of 1968, and this time there was no history of direct trauma to the right eye. Her vision in the right eye was found to be 6/60. Macular edema was present. Pigmentary changes were as before but a fresh perimacular irregular hemorrhage was found. Fluorescein angiography revealed the breaks in Bruch's membrane, and there was further retention of fluorescein in the macular region. The left eye was unchanged.

The final diagnosis was of angioid streaks and traumatic retinopathy secondary to direct or distant trauma (or both), possibly on two separate instances, in January and in August.

Comment This patient is the least typical of the group described in this paper. She certainly harbored an undoubtedly previously established bilateral retinopathy. It is conceivable that her reduction in vision was unrelated to direct injury to either eye. If one discards local trauma, which seems to have been invented by her after the fact, it is possible that she received minimal distal trauma consisting of thoracic or abdominal compression which produced a moderate intravenous pressure elevation resulting in a form of superficial hemorrhagic retinopathy of the right eye.

The episode of recurrence without antecedent trauma again may have represented venous reflux increased pressure due to the Valsalva maneuver, or may be interpreted as a local change in the retina producing a disciform type of degeneration having nothing at all to do with distal stress.

CASE 2

Presented through the courtesy of David Naidoff, MD, of Philadelphia.

This 52-year-old white female was admitted to hospital in December 1970 because of severe anemia and chronic alcoholism. Her admission hemoglobin was less than 4 grams and the hematocrit was 9 per cent. She received four units of blood with supplementary iron and vitamins. A diagnosis of nutritional anemia was established. On discharge in January 1971 her hemoglobin was 9.3 grams and the hematocrit was 29 per cent. The patient stated that, throughout her hospital stay, she had complained of poor visual acuity but that this was ignored. In February 1971 she was seen by an ophthalmologist and her visual acuity was found to be 6/120 in each eye and could not be improved. The rest of the ocular examination was within normal limits except for the fundi which showed deep as well as superficial and preretinal hemorrhages scattered throughout the posterior pole. There was an especially large

preretinal hemorrhage in the region of the macula of the right eye. By March 1971 her vision was 6/9 in the right eye and 6/7.5 in the left eye. Several weeks later this had improved to 6/6 each eye. Except for some pigmentary disturbances and mottled appearance in the macula of each eye the ophthalmoscopic findings were normal. Her family physician reported that her hemoglobin was 14.4 grams and her hematocrit was 45 per cent by this time.

Comment Unfortunately this patient was not examined prior to her ocular complaints. Her underlying systemic condition was a nutritional anemia assumed to have been related to her alcoholism. There is little doubt that all forms of anemia influence capillary permeability in the eye (or elsewhere). Just what type of compression crush injury she may have sustained is unknown, but it is not difficult to imagine this sort of thing occurring during an acute bout of alcoholic digression. Her retinopathy was strictly superficial and hemorrhagic in nature. The changes centered in the macula and were followed by complete recovery. How much was due to trauma remains speculative.

CASE 3

A 36-year-old black male was first seen in the emergency room in June 1971. He had cut the extensor surface of the distal metacarpal of the fifth finger of his right hand with a Christmas tree ornament. This was immediately repaired. Later in June 1971 the patient presented a history of having been hit in the right eye with a fist about 12 hours earlier. During his examination in the eye clinic he stated that he had had poor depth perception at night for some time. Vision in the right eye was 6/7.5 and could be improved to 6/6. Moderate lid edema was present and a subconjunctival hemorrhage was noted. Vision in the left eye was 6/6 and all the findings were normal with the exception of the fundus, which showed irregular peripheral pigmentary migration and accumulations of pigmented clumps centered in both the arterioles and venules, with some areas of gliosis and a few extensive areas of depigmentation in each eye. Fundus photographs were taken and fluorescein angiography was performed. The patient was advised to undergo extensive blood studies by his local internist. Nothing abnormal was found.

The patient was re-examined in July 1971 when his vision had returned to 6/6 in each eye. Repeated fluorescein angiography did not reveal anything new, and a tentative diagnosis of old chorioditis was established. At this time the patient was noted to have limited extension of his previously injured finger and a diagnosis of tendon laceration was established and repair was recommended. Hand surgery was performed on 30 August 1971; it consisted of tendonlysis and tendonplasty of the extensor digitorum communis. Preoperative examination revealed that the patient's blood pressure was 148/90 and the ECG showed slight evidence of left ventricular hypertrophy. Hemoglobin, hematocrit, white blood cell, and differential studies, as well as

urinalysis, were all normal. Anesthesia was induced with sodium pentothal but a reflex spasm of the trachea and bronchial tree developed, and this led to a rather heroic form of tracheal intubation. During the first postoperative day the patient had many complaints, but a sore throat and greatly disturbed vision in each eye were predominant. On 1 September 1971 an orthopedic resident gave him reassurance and permitted him to return home. The next day, 2 September 1971, he returned to the eye clinic where his vision was found to be 6/18 in the right eye and 6/60 in the left eye. Moderate arteriolar narrowing was observed, and large preretinal hemorrhages were present in and over the macula of each eye (Figure 1). These cleared rapidly so that by 7 September 1971 the vision was 6/9 and 6/30. Improvement continued and he was referred to a hematologist who reported only a slight degree of cardiomegaly and low-grade hypertension but no blood dyscrasias. The vision continued to clear gradually over the subsequent months, and by January 1972 it was found to be 6/6 in each eye, with normal visual fields and no other ocular abnormalities except the peripheral pigmentary disturbance (Figure 2).

Comment There was no question in this patient about the direct cause and effect of the hemorrhagic retinopathy. He had a previously photographically documented well established retinopathy, low grade hypertension, and normal vision. He had been exposed to a prolonged Valsalva maneuver and had experienced visual symptoms immediately upon awakening from the anesthesia. Massive preretinal hemorrhages were observed shortly thereafter and these disappeared over a period of a few months.

DISCUSSION

The retinal circulation is an end-artery system and can react in a limited number of ways to distal insults. Either the arterial or the venous circulation is primarily implicated. Most frequently both are involved to varying degrees. This being the case, it seems appropriate to borrow terminology from cardiology where the terms "forward" and "backward" failure have been employed to describe left- and right-sided decompensation.[1] A similar terminology is herein ascribed to retinal vasculopathies as they occur secondary to stress applied at some distance from the head or eye.

It is postulated that retinopathies due to distal trauma are the effects of alterations in the arterial (forward retinopathy) or the venous (backward retinopathy) circulations or to changes concurrently occurring in both systems (mixed retinopathy). Of course, the common denominator and the ultimate site of failure in any of these retinopathies is the capillary circulation.

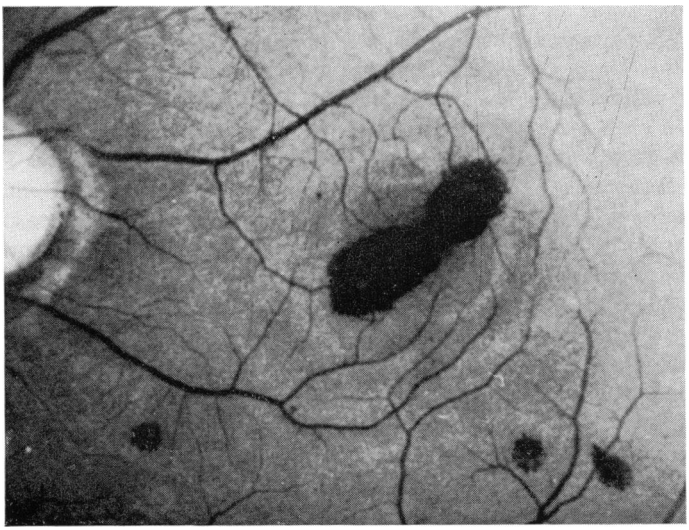

FIGURE 1

Monochromatic green photograph of Case 3 several days after violent Valsalva maneuver. Preretinal hemorrhages centered in the maculae were noted in each eye.

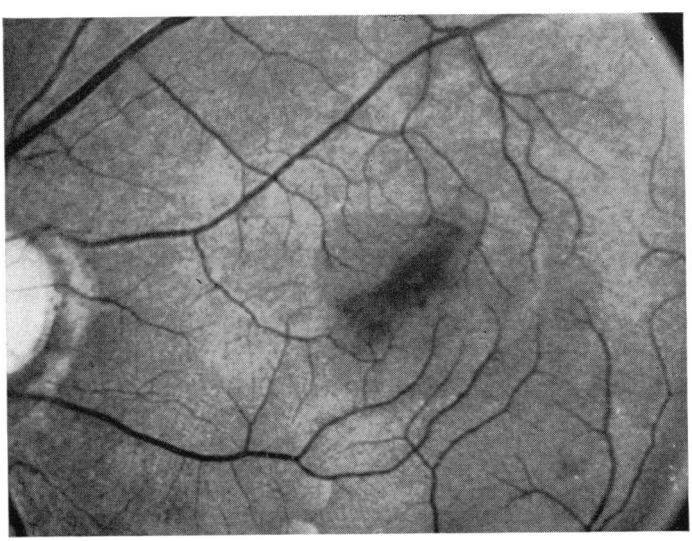

FIGURE 2

Monochromatic green photograph of Case 3 six months later. Hemorrhages have disappeared with no sequelae.

TYPES OF RETINOPATHY DUE TO DISTANT TRAUMA

A. FORWARD – ARTERIAL VASCULOPATHY

Ocular (and cerebral) manifestations of distant trauma are of two types: those due to emboli (fat, air, blood clots), and those due to pressure elevations in the vessels.[2]

Injuries to the skeletal system and the long bones in particular produce fatty emboli which may escape into the venous circulation. If massive, the pulmonary effects of such emboli may prove overwhelming and result in death. In other instances the fat particles, perhaps in solution, circumnavigate the pulmonary circulation and eventually gain access to the left heart whence they may act as arterial emboli primarily producing signs and symptoms in the ocular and cerebral tissues. Histopathologic studies by Jacobi in 1868 showed that these emboli were ether soluble.[3] They have been definitely identified as fat in the uvea and retinal tissues by DeVoe[4] and others, and have been demonstrated in flat preparations of the retina by Kearns.[5] Similar retinal changes can occur from ruptured livers, injected paraffin, or locally from extenteration of the orbit.[6] The question to be answered is how do the fat particles circumnavigate the pulmonary circulation and why are not similar changes noted in triglyceride lipemia retinalis?

The arterial vessels may also respond directly to increased pressure changes. Hypertensive retinopathy is a well recognized entity in ophthalmology. That arterial spasm, striate hemorrhages, edema, and possibly even cotton wool spots can arise secondary to sudden increased arterial pressure has been less well appreciated. The hydrostatic pressure syndrome producing retinal changes has been documented by Byrnes.[7] Both transverse G and negative G, besides their more obvious effects on the venous system, produce increased pressures within the arterial system. The resulting ocular changes depend upon the rapidity of onset, the magnitude of the force, and the duration of exposure. Finally, especially during transverse G exposures such as occur in linear acceleration, it is impossible to eliminate direct forces pushing on the globe.

B. BACKWARD – VENOUS VASCULOPATHY

The venous system rostral to the heart has little need for intraluminal valves since gravity, under usual circumstances, guarantees adequate cardiac return. Therefore any rise in the intrathoracic or intra-abdominal pressures particularly against a closed glottis (coughing, vomiting, lifting, straining at stool, wind blast injuries, hydrostatic column syndrome, and especially crush or compression injuries) are all expressions of the

well known Valsalva maneuver which produces a reflux venous pressure rise in the upper reaches of the body.[8]

Such venous pressure elevations, particularly if they are sudden in onset, produce many signs, including subconjunctival hemorrhages, skin petechia in the hand and neck, the bluish cyanotic mask syndrome, and traumatic asphyxia.

The fundi may escape unscathed even when vision has been diminished if the injury is one of sustained pressure not associated with sudden onset. Just the opposite is true for the more common sudden and massive Valsalva maneuver. Here, depending upon the magnitude of the compressive force and the previous state of the retinal vessels, the ophthalmoscopic appearances vary from isolated, barely recognizable, localized edema to massive "fluffy clouds" of superficial (inner retinal) edematous transudates or, at worst, to massive superficial and preretinal hemorrhages.

Examples of the latter – Valsalva hemorrhagic retinopathy – are described above in the case reports. Similar findings have been noted by others. Walsh and Hoyt analyzed the report of a gymnast who fell from a horizontal bar[9] and Hoyt reported another patient who had being suspended by the head during pneumoencephalography.[10] Both of these presented similar ophthalmoscopic pictures. In a personal communication Hoyt has further described a boy who was doing bench presses with 175-pound weights, a girl water skier who held her breath as she was flipped by a wave, and a lady who was helping a neighbor move her refrigerator down a flight of stairs. All of these patients had a variable but pure hemorrhagic retinopathy, most of which was preretinal and on occasion even extended into the vitreous.[11]

C. MIXED – FORWARD ARTERIAL AND BACKWARD VENOUS VASCULOPATHY

It is a contention of this report that what has formerly been called Purtscher's retinopathy[12,13] due to distant trauma is in reality a mixed state of affairs.

Multiple injuries in or about the long axis of the body are frequently accompanied by crush or compression forces, broken bones, and liver damage. The effects on the retina are the outcome of both the arterial and venous insults. These in turn depend upon the duration of the force, the position of the long arterial and venous systems at the time of injury, the rate of onset, the magnitude and direction of the forces, and the previous status of the retinal circulation (presence or absence of disease).

Thus Purtscher's retinopathy may be accompanied both by deep hard exudates, striate hemorrhages, and arterial spasms, representing forward

TABLE 1. PATHOPHYSIOLOGY OF RETINOPATHIES DUE TO DISTANT TRAUMA

I FORWARD – ARTERIAL VASCULOPATHY
- A. Fatty embolization: hard yellow-white retinal exudates
- B. Negative G (arterial pressure rise): arterial angiospasms
- C. Transverse G (arterial pressure rise): striate hemorrhages

II BACKWARD – VENOUS VASCULOPATHY
- A. Valsalva maneuver (venous pressure rise)
  1. Mild: retinal edema
  2. Moderate: superficial transudates "fluffy clouds"
  3. Severe: preretinal hemorrhages
     a. Valsalva hemorrhagic retinopathy (previously diseased retina)

III MIXED (PURTSCHER'S RETINOPATHY) FORWARD AND BACKWARD VASCULOPATHY
- A. Forward: fatty emboli, yellow-white exudates, arterial spasm, striate hemorrhages
- B. Backward: localized edema, fluffy transudates, preretinal hemorrhages
- C. (Direct-Berlin's Commitio retinopathy, choroidal and retinal tears, edema, hemorrhages)

changes, and also by mild retinal edema, fluffy retinal edema, and preretinal bleeding, representing backward changes. In attempts to explain pathophysiology there seems little need to implicate extrusions of the cerebrospinal fluid, and it seems equally fallacious to ascribe all the changes to observed angiospasms or histopathologically proved fatty embolization. In addition most of these patients have been subject to massive crush injuries, and as a result are much more likely to have been exposed to direct blows upon the globe (see Case 1) which further complicate interpretation of retinal findings.

A summary of the various combinations of postulated pathophysiologic mechanisms is found in Table 1.

The literature on this subject has been well reviewed by Marr and Marr[14] who have brought order out of chaos regarding understanding of ocular findings. The world literature on ocular and systemic effects in the central nervous system and elsewhere as they occur following distant trauma have been summarized by Duke-Elder,[15] and more recently by Walsh and Hoyt.[16] Their writings have served as a foundation for this report.

SUMMARY

A particular form of retinopathy, preretinal and hemorrhagic in nature, and secondary to a sudden increase of intrathoracic pressure (against a closed glottis), the well known Valsalva maneuver, has been described.

In an attempt to understand the pathophysiology involved a new scheme for understanding retinopathy due to distant trauma has evolved. Three categories are postulated: (1) forward or arterial alterations including fatty emboli and arterial spasm; (2) backward or venous alterations including small and large transudates and hemorrhages; and (3) mixed – a combination of forward and backward changes. The various features heretofore described under the heading of Purtscher's retinopathy are here considered to be manifestations of the third type, the mixed forward and backward retinopathies. Thus the numerous and inconsistent previously described ophthalmoscopic appearances are placed on a feasible basis.

REFERENCES

1. Friedberg, C.K., Diseases of the Heart, 3rd ed., Philadelphia, Saunders, 1966, pp. 270–3.
2. Walsh, F.B., and W.F. Hoyt, Clinical Neuro-Ophthalmology, 3rd ed., Baltimore, Williams & Wilkins, 1969, III, 2454–7.
3. Jacobi, J., Casuistische Beitrage: Part IV – Ophthalmoscopischer Befund bei Fractura Basis Cranii, Graefe Arch. Ophthal., 14:147, 1868.
4. DeVoe, A.G., Ocular fat embolism: A clinical and pathological report, Trans. Amer. Ophthal. Soc., 47:254, 1949.
5. Kearns, T.P., Fat embolism of the retina, Amer. J. Ophthal., 41:1, 1956.
6. Duke-Elder, S., Textbook of Ophthalmology: Volume VI – Injuries, St Louis, Mosby, 1954, p. 6374.
7. Byrnes, V.A., Elevated intravascular pressure as an etiologic mechanism in the production of eye injuries, Trans. Amer. Ophthal. Soc., 57:473, 1959.
8. Duke-Elder, S., Textbook of Ophthalmology: Volume VI – Injuries, St Louis, Mosby, 1954, pp. 6375–8.
9. Walsh, F.B., and W.F. Hoyt, Traumatic retinal angiopathy; Purtscher's disease; retinopathy from sudden elevation of intravascular pressure in the head and eye; types of injuries without overt damage to the chest, in Clinical Neuro-Ophthalmology, 3rd ed., Baltimore, Williams & Wilkins, 1969, III, 2445, 2447.
10. Hoyt, W.F., and D. Beeston, The Ocular Fundus in Neurologic Disease, St Louis, Mosby, 1966, p. 119.
11. Hoyt, W.F., personal communication.
12. Purtscher, O., Noch unbekannte Befunde nach Schaedeltrauma, Ber. Versamml. Ophthal. Ges. Heidel, 36:294, 1910.
13. Purtscher, O., Angiopathia Betinae Traumatica: Lymphorrhagien des Augengrundes, Graefe. Arch. Ophthal., 82:341, 1912.
14. Marr, W.G., and E.G. Marr, Some observations on Purtscher's disease: Traumatic retinal angiopathy, Amer. J. Ophthal., 54:693, 1962.
15. Duke-Elder, S., and P.A. MacFaul, System of Ophthalmology: Volume XIV, Part 1 – Mechanical Injuries: The Ophthalmological Implications of Remote Injury, St Louis, Mosby, 1972, pp. 716–43.
16. Walsh, F.B., and W.F. Hoyt, Ocular and cerebral signs of remote trauma, in Clinical Neuro-Ophthalmology, 3rd ed., Baltimore, Williams & Wilkins, 1969, III, 2433–57.

DISCUSSION

DR A.G. DEVOE. In 1949 I presented before this Society a report of a 25-year-old man who had been injured in an automobile accident but who remained fully conscious, alert, and cooperative despite multiple compound, comminuted, and simple fractures of both tibias and fibulas, a fracture of the eleventh rib on the right, and fractures of the transverse processes of the first to the fourth lumbar vertebrae. There was no direct injury to the head. He remained in good condition until 20 hours past injury when he suddenly became comatose with a rapid fleeting pulse, proceeding to a deep coma with Cheyne-Stokes respiration. Upon admission to our hospital he was cyanotic with skin covered with petechiae. Numerous petechial hemorrhages were present in the palpebral and bulbar conjunctiva of both eyes. His course was continuously downhill, death ensuing 36 hours after admission to the hospital and two and a half days after the original injury. Eighteen hours prior to death the eyes were examined; petchial hemorrhages were noted in the conjunctiva bilaterally but there was nothing to suggest local injury. The disks were normal, and there was no detectable change in the retinal vasculature. At the macula a typical cherry red spot, surrounded by an area of edema, was noted. Adjacent to this were six discrete fluffy exudates of the fresh cotton wool type. No hemorrhages were noted.

A complete autopsy demonstrated venous engorgement and fresh interstitial hemorrhages in all organs. When stained with scarlet red, massive fat embolization of the capillaries and venules in all organs as well as fat globules within the alveoli of the lungs and lumens of the renal tubules were seen. Throughout the choroid and ciliary processes globules of fat were irregularly encountered in the vasculature. The retina also presented numerous vessels occluded with fat. These were much more obvious at the posterior pole but were also found peripherally.

This patient probably had the typical retinopathy associated with distant trauma. Dr Duane has pointed out that it is but one of the numerous ways in which the retina can be affected by trauma not applied directly to the eye. Purtscher's retinopathy, characteristically seen in sudden chest compression, is a hydrostatic phenomenon accompanied by exudates and hemorrhages in the retina; it is usually not lethal, nor accompanied by serious visual loss. The hydrostatic pressure syndrome occurring in rapid deceleration, as in ejection from a supersonic plane, is usually associated with congestion of the head, face, and conjunctiva as the blood is displaced peripherally under the force of gravity. The retinal picture is usually one of hemorrhage. Fat emboli, on the other hand, represent a mechanical obstruction due to globules of fat derived from broken bones. These pass through the lungs because of defective pulmonary circulation associated with falling peripheral blood pressure and enlargement of the right side of the heart. Emboli reach all organs of the body, and the condition is frequently followed by death.

Undoubtedly the Valsalva type of retinopathy can occur, but as I read the histories of Dr Duane's patients it did not seem to me that sufficient evidence to establish the diagnosis could be obtained. In Case 1, angioid streaks were present and there seems to have been active retinopathy as evidenced by the recurring fresh hemorrhage eight months after the original injury. The patient also had a previously established bilateral retinopathy prior to the alleged acute episode. Case 2 had severe anemia with a hemoglobin of less than 4 grams and a hematocrit of 9 per cent. This in itself could be sufficient to cause hemorrhages and exudate. There was no definite history of any compression or crush injury, merely the fact that she was a known alcoholic. In Case 3 a definite local injury with a blow from a fist was recorded. The acute retinitis, noted eight months later, presumably followed laryngospasm associated with endotracheal intubation during general anesthesia. This is not too uncommon an occurrence in a hospital performing many general anesthetics. I have not examined the fundus of such an individual on the table, but in view of the history that Dr Duane has presented it might be worthwhile for us to do so. It seems quite conceivable that sufficient back pressure could be built up to produce the type of hemorrhages so frequently seen in the newborn.

Dr Edward W.D. Norton. I would like to show three cases.

[Slide] This is a 33-year-old black male. This man was perfectly well, as far as we know, with no problems, until about 3 weeks before this photograph was taken. He awoke one morning with a headache. He had been at a party the night before. He vomited, and when he vomited he suddenly noticed loss of vision in his right eye. These photographs were taken 3 weeks later, as I said. At the time we took these photographs his vision was at the level of around 20/100.

[Slide] Over the next 3 months the hemorrhage on the right-hand side, which I think is an infraretinal, subinternal, limiting-layer retinal hemorrhage – and I don't think they are preretinal in the sense that they are between the internal limiting layer and the vitreous, but that is another matter – never broke into the vitreous, and in the course of 3 months it cleared to the level of the right eye, in which there is 20/20 vision.

[Slide] This is a young woman of 27 in her eighth month of pregnancy, perfectly well, who awakened at 8 am one day with a coughing spell. She lay on the bed and coughed. When she lifted her head she could not see out of this eye. She came to see me, and these pictures were taken a few hours later. You can see in the upper left-hand corner that she has a preretinal hemorrhage which has broken into the vitreous. Her vision was at the 20/400 level. The bottom right-hand picture was taken about 3 months later, at which time her vision was back to 20/20. I could not find her picture a year later, but she had a little pigment change. It was only a coughing spell.

[Slide] This is a very interesting case. This is a 33-year-old physician, unmarried, who was with his girl friend. I don't know all of the details, since he was applying for an ophthalmology residency, but anyway he did admit that

at 3:30 am he and his girlfriend had had a rather violent argument, and he said it led him to such a fit of rage that he could not control himself, and suddenly he realized the vision in his eye had gone. You can see the massive hemorrhage he has, which is mostly preretinal. Notice some of the hemorrhages along the venous channels. They show up very well in the angiogram. His vision was down to counting fingers at this point. He has subsequently recovered completely, to 20/20 vision. The only thing remaining is that some of the blood had broken through into the vitreous and you can see it inferiorly.

Actually I think there is such an entity as Dr Duane has proposed. I don't think it required an abnormal vascular system. I personally think these people probably had a sudden rise in intravenous pressure and the vessels broke down and gave rise to these hemorrhages.

DR THOMAS P. KEARNS. [Slide] This first patient is a boy 15 years of age who awoke on a Sunday morning with this picture. He had some macular edema, but the main pathology is the preretinal hemorrhage. He was referred to us because the ophthalmologist suspected he had leukemia. I must admit that this was my thinking too.

We found from the history that this boy had been camping Saturday night with a friend, and they were horsing around, as 15-year-old boys are apt to do. We then wondered if the hemorrhage was due to direct trauma. If you listen to the patient, he will sometimes give you the diagnosis, and that is what happened here. He said, "I was blowing up an air mattress. Do you think that had anything to do with this?" So we called this an "air mattress retinopathy." I think the term "hemorrhagic Valsalva retinopathy" is much better.

[Slide] This was a young man of 22, a clerk in a department store in our city. He awoke on a Monday morning with blurred vision in his left eye. I thought that the preretinal hemorrhage was caused by leukemia or anemia or some other serious systemic disease, and I was all set to send him for more tests. He said, "That's going to cost money."

I said, "Yes, but you don't know what trouble you may have."

He said, "Doc, I think I'll wait on those tests, because I really hung one on Saturday night before this happened. Do you know what the dry heaves are?"

I allowed I had heard of them. Then I realized this was a "dry heaves retinopathy."

There are two points I would like to make. First, I agree with Dr Norton that this does not have to be a diseased retina. Both of my young males certainly had normal retinas. Second, as Dr Norton said, I think the venous back pressure is the mechanism involved here.

I would like to continue to use the term "hemorrhagic Valsalva retinopathy." I think this is a real contribution. Thank you.

DR J. REIMER WOLTER. I agree with all the basic conclusions that Dr Duane

has made, but I would like to take issue with a minor detail, and I feel obligated to do so because of my earlier interest in this subject matter.

I believe that fat embolism does not primarily cause deep exudates in the retina. Fat embolism typically causes cotton wool spots due to microinfarction of small zones of inner retina. This results in local edema, interruption of nerve fibers with axonal enlargement, and, sometimes, minute hemorrhage in the nerve fiber layer. Deep hard exudates, in contrast, are a result of chronic leakage of retinal blood vessels and they are accumulations of a combination of lipids with proteinaceous fluids in the outer retinal layers. I have never seen deep hard exudates in the retina as a direct result of fat embolism.

Dr George N. Wise. In Purtscher's retinopathy there are three things that are rather difficult to explain on the basis of fat embolization from traumatized bones. First, as exemplified in Dr DeVoe's case, is the number of retinal emboli present in some fundi. The number of cotton wool spots greatly exceeds the number of emboli one might expect from the degree of bone damage in some patients, especially as retinal emboli probably represent only a small part of the total embolization. Second, embolization by liberated bone marrow should occur promptly and there is usually a delay of one or more days in the onset of Purtscher's retinopathy following the injury. Third, any embolization from traumatized bones should be filtered out in the capillary bed of the lungs.

There is a more recent explanation for the vascular blockage in Purtscher's retinopathy that is of interest, although not yet fully proven. Rather than bone marrow embolization, it has been suggested that a physiochemical change in the size of the chylomicrons, produced in some way as a result of the trauma, induces macromolecules large enough to block small vessels. Such an explanation, if correct, would answer all of the three objections cited above.

In regard to the preretinal hemorrhages, it is usually those involving the macular area which bring the patient to the physician. I have never seen a preretinal macular hemorrhage occurring spontaneously in a young, otherwise healthy individual which did not clear, with full return of macular function. I wonder if anyone here has seen such a hemorrhage result in a permanent visual defect.

Dr Harold F. Falls. I wish to comment about three cases in which subsequent information aided somewhat in ascertaining the etiology.

In Michigan schools most of the children participate in the "Decathlon" – competing in push-ups, sit-ups, chinning, and so on. One young man complained of visual loss following completion of 65 push-ups. We did not see him until one week later, but even then he still presented bilateral superficial macular hemorrhage. This patient volunteered the information that a 19-year-old male cousin had experienced "hemorrhages in his eyes" while participating in obstacle exercises during marine training. Subsequent information revealed a mild capillary fragility defect in both young men.

In consultation I recently saw a middle-aged female who had experienced "a stormy anesthesia." She presented a spittle or frothy superficial hemorrhage in her left macula.

All three individuals eventually recovered normal vision without detectable retinal change.

These cases could conceivably fall into Dr Duane's classification.

MR STEPHEN MILLER. The hemorrhages that have been shown this morning remind me of the hemorrhages that occur following subarachnoid hemorrhage, where you get a sudden rise of cerebrospinal fluid pressure, and then the veins give way even though they are healthy. It may be that the mechanism of these preretinal hemorrhages is not a direct one, due to a rise in venous pressure with no valves affecting the retinal veins. It may have come indirectly from the rise in cerebrospinal fluid pressure, and secondly this is transmitted to the veins.

What makes one think this is more than possible is that the recent work of Hayreh, on the blood supply to the nerve head, shows that there are no ciliary veins corresponding to the posterior ciliary arteries; in fact, all the blood from the macular area has to go through the central retinal vein, and this would make it particularly liable to give way if the cerebrospinal fluid pressure suddenly rose.

DR DUANE. I am pleased that there has been so much discussion of a non-surgical subject.

I thank Dr DeVoe for his remarks. He is absolutely correct, that the fat particles do collect in the lungs. I am not sure getting from the right to the left side is fully explained, the way he described it, but Sudan studies of sputum in patients who do develop cephalad types of difficulties, including ones in the eye, have been recognized over the years.

I also don't know precisely what happens in judo, but I do know that one of the tricks is to be thrown and to hit the floor with a sudden compression of the thoracic chest; this is not unusual. Looking at "postmortems" of types of distant retinopathy that have been described – the original one by Purtscher – and at other patients described in the literature, it is always suggested that possibly a Valsalva type of maneuver has occurred, as an explanation of the changes that have been observed.

I am glad Dr DeVoe also brought up the patient with anemia. I admit the first case in my presentation leaves one pretty far out to explain Valsalva hemorrhagic retinopathy, but I forgot to mention in my main talk that all of us see hemorrhages in patients who have blood dyscrasias, particularly severe anemia. It is not easy to explain the hemorrhage. Most of these eyes, or at least many of them, do not come to postmortem examination.

I think it is not unlikely that many of these people have had various types of Valsalva maneuvers – straining at stool, coughing, bending, all those things that we ask cataract patients not to do. It is not inconceivable that minor

trauma of that sort, which would not produce any changes in normal vasculature, will do it in a person who is highly anemic.

I agree also with Dr Norton that most of these hemorrhages are beneath the internal limiting membrane of the retina, although, as he himself has seen, some of these do break through into the vitreous.

Over and above the cases that he has described, Dr Hoyt had seen a copy of this paper and he described a whole series of similar cases to me. He said in particular he wanted to refer to his fundus atlas, wherein he presented a patient who had had pneumoencephalography and where the patient had been almost hanged on the head halter. I looked it up and, sure enough, there is a picture just like the ones Dr Norton showed. Hoyt also said he saw it occur in a young boy doing bench presses, in a girl water skier who held her breath when she flipped into a wave, and in a neighbor who was helping a lady move a refrigerator down a flight of stairs.

Dr Wolter commented regarding fat embolism. I am not enough of an ocular pathologist to know the nuances of that, although I do recall in Dr DeVoe's paper that he showed these emboli many places besides the superficial retina, namely, all through the choroidal circulation and into the ciliary body.

Dr Reese, I probably skipped over it rapidly, but one of the theses developed in the paper was that there are no valves in the cephalad portion of the vascular system, and therefore pressure might be transmitted more readily in that direction than inferiorly, although I don't know that that really holds up either, because with positive G exposure, where blood is driven to the lower ends of the body, legs swell. I don't think the valves in the veins are very firm anyway.

I am pleased to hear what Dr Wise had to say about fat emboli. I did see the reference in his new book, which Dr Cogan calls "the product of the three wise men," and just what these chylomicrons are, and how they get through the circulation, and how they are different from liver emboli or various other types of emboli, still remains to be explained. One opens up many more questions than he answers in this type of study.

I am glad Dr Falls pointed out that vision usually returns in these patients. Coming back to what Dr DeVoe said, I agree that, whether patients have symptoms or not, those that have had rough types of anesthesia should be examined for medico-legal purposes. Fortunately, whether hemorrhages are perimacular or macular, it has been my experience also (and I think the experience of most people to whom I have spoken) that normal vision does return.

I think Dr Falls is kind of listening to the grass grow if he says it is more likely to occur in the left eye than in the right eye. That is certainly true for emboli, but much of this phenomenon – certainly the hemorrhagic retinopathy – is on the basis of the venous changes, and therefore I don't think one need postulate a different type of anatomical system such as we do for arterial phenomena.

Regarding the influence of cerebrospinal fluid, I suppose all of these types of retinopathy are cousins. We have come almost full circle. Purtscher in 1910 and later in 1912 thought the retinopathy that he described was due to a rise in pressure in the vaginal sheath pushing the cerebrospinal fluid through the optic nerve and into the eye. I know that is not proposed here, of course. But even if it is shown that the vascular system is quite different from the arterial system in and about the papillary region, if one postulates that the central retinal vein is implicated, it terminates pretty much in the capillary regions; that is, many of the capillaries are in the macular area, so I think it could still be due to pressure on the central vein and still be expressed in the macula.

Of course this is an armchair philosophy, and I think the subarachnoid type of retinopathy that we all see is a variation of another form of distant retinopathy.